INTRODUCTION

In July 1991, P.N. Lee prepared a first report on the role of confounding variables in the relationship between ETS and lung cancer. That report, inter alia, listed a number of factors known or thought to be a cause of lung cancer, subdividing them into those considered, definite, probable and possible causes of lung cancer and those considered probably not a cause of lung cancer. That review only gave relatively cursory supportive evidence for this belief.

While not intended as a full and comprehensive review of the evidence for each factor, this report considers each factor in rather more detail. The intent was to prepare, for each factor, a brief summary of the key evidence, giving enough detail for the reader to be able to gain an insight into the consistency and strength of the evidence and the likely magnitude of any association with lung cancer. No attempt has been made to do a full literature search for evidence on each risk factor. Major sources of information have been the IARC Monographs and P.N. Lee's quite extensive files on lung cancer risk factors accumulated over many years. When reading available papers revealed the files missed important other papers, copies of these were obtained and considered. However, no claims are made that all relevant papers have been considered.

The major part of the work was carried out by A.J. Thornton who is leaving epidemiology for ecology at the end of June 1994. Her impending departure has meant a few risk factors could not be covered in the planned style. Nevertheless, the report should prove a valuable reference work.

THE RISK FACTORS

1. Acrylonitrile

Table 1 details the studies found which attempted to relate lung cancer risk to exposure to acrylonitrile. The six standardized mortality ratios calculated ranged from 100-400, with four of them being raised.

The main drawback of the studies is that none of them appeared to have taken any objective measurements of the levels of acrylonitrile the subjects were exposed to. It is obvious, therefore, that inaccuracies may have been introduced. Not surprisingly, in view of this lack of information, IARC considered the evidence for the carcinogenicity of acrylonitrile to humans to be "limited" [2].

Table 1: Estimates of standardized incidence ratio for exposure to acrylonitrile

Study	Population	Standardized mortality ratio
US Dept of Labor (1978)[1]	US male textile fibre plant workers	400[2]
Waxweiler et al (1981)[3]	US rubber workers	100
Werner and Carter (1981)[3]	UK male polymerization workers	118[2,4]
Delzell and Monson (1982)[3]	US rubber workers	153[2]
O'Berg et al (1985)[3]	US male textile fibre plant workers	139[2]
Chen et al (1987)[3]	US textile fibre plant workers	100

1 From International Agency for Research on Cancer (1979)

2 Estimated from data given

3 From International Agency for Research on Cancer (1987)

4 Tumours of the respiratory system

References

1. International Agency for Research on Cancer (1979) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 19: Some monomers, plastics and synthetic elastomers, and acrolein, 73-113. IARC, Lyon.

2. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 79-80. IARC, Lyon.

2. Adverse life situations

Table 2 gives details of those studies found which attempted to relate the risk of lung cancer to having previously suffered from an adverse life experience, such as divorce, bereavement, unhappy home or similar situations. One relative risk was estimated, of 2.73, along with two standardized incidence ratios, of 176 and 214. One study reported that adverse life situations, both in childhood and adulthood, occurred more frequently in lung cancer cases than in controls, while another reported that smokers with lung cancer were more likely to have been severely shocked by war experiences than smokers without lung cancer. Finally, one study observed an increasing gradient in lung cancer mortality across groups of single, married, widowed and divorced men.

However, it should be noted that some inconsistencies were found. For example, in the studies by Kissen childhood parental bereavement appeared to be associated with lung cancer risk, while sibling bereavement and adult bereavement of a close relative were not. Thus, while the findings of the studies are suggestive of an influence of adverse life situations on the risk of lung cancer, the evidence is not particularly strong.

Table 2: Estimates of relative risk for adverse life situations

Study	Population	Relative risk (95% limits)
Smith (1966)[1]	Scottish men aged 55-64	Gradient in lung cancer mortality increased across single, married, widowed and divorced men
Kissen (1967)/ Kissen (1969)	Male patients aged 55-64	Adverse life situations occurred more frequently in lung cancer cases than in control group
	As above	Adverse life situations in childhood occurred twice as frequently in lung cancer cases than in control group
Blohmke et al (1981)	German men	Smokers with lung cancer had more frequently been severely shocked by war experiences than smokers in control group

Table 2 continued

Jones et al (1984)	UK residents	Standardized incidence ratio of 176 (62-350) estimated for subjects widowed before 1966
	As above	Standardized incidence ratio of 214 (38-533) estimated for subjects widowed between 1966-68
Liu et al (1993)	Chinese residents	Relative risk of 2.73(1.34-5.64) found for those suffering "serious psychic trauma"

1 From Kissen (1969)

References

1. Blohmke M, Engelhardt BV and Stelzer O (1981) Investigations on the personality of patients with pulmonary carcinomas compared to a control group. Med Biol Environ, 9, 67-75.

3. Jones DR, Goldblatt PO and Leon DA (1984) Bereavement and cancer: some data on deaths of spouses from the longitudinal study of Office of Population Censuses and Surveys. Br Med J, 289, 461-464.

4. Kissen DM (1967) Psychosocial factors, personality and lung cancer in men aged 55-64. Br J Med Psychol, 40, 29-43.

5. Kissen DM (1969) The present status of psychosomatic cancer research. Geriatrics, 24, 129-137.

6. Liu J-Z, Hu H-S, Hu Y-H, Lu P and Hao L-Y (1993) A study on the relation between indoor exhaust and lung cancer. Proc Indoor Air, 1, 489-492.

3. Air pollution

Although many studies have attempted to estimate potential risks between lung cancer and specific pollutants, several have also considered general air pollution, and details of these are given in Table 3. Thirty-six relative risks were presented from cohort studies, ranging from 0.95-2.50. Only four risk estimates did not exceed 1.00. Estimates of the percentage relative differences in mortality or morbidity between various populations were also presented by some of the studies, and these ranged between 0-405%. Additionally, one study estimated that up to 40% of lung cancers were caused by urban factors other than smoking. For the case-control studies the estimates of relative risk ranged from 1.00-4.0, with 18 out of 19 being raised.

The major drawback with these studies is that only seven [5-8,12,14,16] made objective measurements of air pollution levels, with four others [1,3,4,13] relying instead on markers such as place of residence, and the remainder failing to give any details. Thus, it is obvious that inaccuracies may have been introduced. Additionally, as air pollution is a complex mix of chemicals, the actual exposures will differ from study to study, depending on the source of the pollution, and it will be impossible to separate out the effects of any one compound.

There is also the further problem of confounding by the effects of tobacco smoking, as it has been suggested that differences exist between the smoking habits of urban and rural dwellers [1,3,6,9,10,12-15]. Although most of the studies gathered data on the respondents' smoking habits, and carried out some type of adjustment during their analyses, two studies (Ehrenberg, Ponka) did not, and, in a further 10 studies (Pershagen and Simonato, Clemmesen and Nielsen, Hoffman and Gilliam, Griswold, Mancuso, Eastcott, Dean 1959, Levin, Mills, Xu) it was not possible to tell whether adjustment had been carried out due to insufficient information being given.

Therefore, although some of the studies do appear to show a higher risk of lung cancer in urban populations, it is not clear how much of this effect is due to air pollution. It has been stated that "urban air contains carcinogenic compounds, but the relatively small excess risk to men occupationally exposed to large concentrations of these compounds raises doubt about the relevance to lung cancer of the much lower levels

found in the air of even the most polluted city" [11]. It is not altogether surprising, then, that the possibility of a link between air air pollution and cancer has been described as "questionable", with any risk, if it does exist, "likely to be extremely small" [2].

Table 3: Estimates of relative risk for exposure to air pollution

Study	Location	Relative risk (95% limits) Male	Female
Cohort studies:			
Clemmesen and Nielsen (1952)[1]	Denmark	405[2]	40
Hoffman and Gilliam (1954)[1]	USA	81[3]	29
Griswold et al (1955)[1]	Connecticut, USA, 1941-46	68[2]	41
	1947-51	75[2]	24
Mancuso et al (1955)[1]	Ohio, USA	78[3]	-
Eastcott (1956)[1]	New Zealand	--51[3]--	
Hammond and Horn (1958)[4]	USA	1.33	-
Dean (1959)[1]	South Africa	--16[3]--	
Levin et al (1960)[1]	New York State, USA	92[2]	33
Mills (1960)[1]	Cincinnati, USA	177[3]	0
Hammond (1962)	USA	1.36	-
Buell et al (1967)[4]	California, USA	1.30	-
Cederlof et al (1975)[4]	Sweden - city dwellers	1.60	Similar
	Town dwellers	1.20	trend
Lloyd (1978)	Scotland	2.5 ($p<0.001$)	-
Hammond and Garfinkel (1980)	USA, large metropolitan area	1.11[5]	-
	Small metropolitan area	1.10[5]	-
	Non-metropolitan area	0.95[5]	-
Doll and Peto (1981)[4]	UK	--1.00--	
Ehrenberg et al (1985)[4]	Sweden	40%[6]	20%

Table 3 continued

Xiao and Xu (1985)	Shen Yang, China	--1.47(p <0.05)--	
Tenkanen and Teppo (1987)[4]	Finland	1.20	-
Pershagen and Simonato	Miyagi, Japan	1.1	1.1
(1990)	Slovakia, Czechoslovakia	1.0	1.4
	Saarland, FRG	1.2	1.3
	Calvados, France	1.2	1.2
	Doubs, France	1.4	1.7
	Szabolcs, Hungary	1.2	1.7
	Norway	1.6	1.9
	Cluj County, Romania	1.0	1.4
	Vaud, Switzerland	1.1	1.6
	England and Wales	1.3	1.3
	New South Wales, Australia	1.2	1.5
Ponka et al (1993)	Helsinki, Finland	1.08[5]	1.86
Case-control studies:			
Stocks and Campbell (1955)[4]	North Wales/Liverpool	--1.1-3.4--	
Haenszel et al (1962)[4]	USA	1.43	-
Haenszel and Taeuber (1964)[4]	USA	-	1.27
Dean (1966)	Northern Ireland	3.52[5]	-
Hitosugi (1968)[4]	Osaka, Japan	--1.2-1.8--	
Dean et al (1977)[4]	North-east England	1.70	1.60
Dean et al (1978)[4]	North-east England	2.30	1.40
Vena (1982)	Erie County, USA	1.09	-
		(0.66-2.20)	
Ulmer (1982)	Ruhr, Germany	--1.57 (p<0.05)--	
Samet et al (1987)[4]	New Mexico, USA	--1.00--	
Xu et al (1989)	Shen Yang, China	2.3	2.5
		(1.7-2.9)	(1.8-3.5)
He et al (1990)	Xuan Wei, China	4.0	3.7
Jedrychowski et al(1990)	Cracow, Poland	1.48	1.17
		(1.08-2.01)	(0.7-1.96)
Katsouyanni et al (1991)	Athens, Greece	-	2.13[5]
			(0.92-4.91)

Footnote to Table 3

1 From Wynder and Hammond (1962)

2 Percentage relative difference in morbidity between study populations

3 Percentage relative difference in mortality between study populations

4 From Pershagen (1989)

5 Estimated from data given

6 Percentages of cancers due to "urban factors"

References

1. Dean G (1966) Lung cancer and bronchitis in Northern Ireland. Br Med J, 1, 1506-1514.

2. Godlee F (1991) Air pollution: II - road traffic and modern industry. Brit Med J, 303, 1539-43.

3. Hammond EC (1962) Air pollution, smoking, and health. Texas J Med, 58, 639-647.

4. Hammond EC and Garfinkel L (1980) General air pollution and cancer in the United States. Prev Med, 9, 206-211.

5. He X, Chapman RS, Yang R, Cao S, Mumford JL and Liang C (1990) Lung cancer and indoor air pollution in Xuan Wei, China: current progress. In: Kasuga H (ed.) Indoor Air Quality, 435-441. Springer-Verlag.

6. Jedrychowski W, Becher H, Wahrendorf J and Basa-Cierpialek Z (1990) A case-control study of lung cancer with special reference to the effect of air pollution in Poland. J Epidemiol Comm Health, 44, 114-120.

7. Katsouyanni K, Trichopoulos D, Kalandidi A, Tomos P and Riboli E (1991) A case-control study of air pollution and tobacco smoking in lung cancer among women in Athens. Prev Med, 20, 271-278.

8. Lloyd OL (1978) Respiratory-cancer clustering associated with localised industrial air pollution. The Lancet, Feb 11, 318-320.

9. Pershagen G (1989) Air pollution and cancer. Published in IARC Monograph series?

10. Pershagen G and Simonato L (1990) Epidemiological evidence on air pollution and cancer. In: Tomatis L (ed.) Air pollution and human cancer, 63-74. Springer-Verlag.

11. Pike MC, Gordon RJ, Henderson BE, Menck HR and SooHoo J (19??) Air pollution. In: Fraumeni (ed.) High risk groups and cancer, 225-238. (Incomplete reference)

12. Ponka A, Pukkala E and Hakulinen T (1993) Lung cancer and ambient air pollution in Helsinki. Environ Int, 19, 221-231.

13. Ulmer WT (1982) Bronchial cancer in cities and rural districts in relation to smoking habits: epidemiological study on the city of Bochum. Inn Med, 9, 410-416.

14. Vena JE (1982) Air pollution as a risk factor in lung cancer. Am J Epidemiol, 116, 42-56.

15. Wynder EL and Hammond EC (1962) A study of air pollution carcinogenesis I: analysis of epidemiological evidence. Cancer, 15, 79-92.

16. Xiao H-P and Xu Z-Y (1985) Air pollution and lung cancer in Liaoning Province, People's Republic of China. Natl Cancer Inst Monogr, 69, 53-58.

17. Xu et al (1989) No reference given.

4. Alcohol

Although lack of time meant that a full evaluation of the possible association between lung cancer risk and alcohol drinking could not be presented in this document, the subject was reviewed by IARC in 1988 [1]. Their evaluation was based on fifteen cohort studies of alcoholics, of persons with higher than average consumption of alcoholic beverages and of the general population, and five case-control studies. The cohort studies yielded inconsistent results on the association between the drinking of alcoholic beverages and the risk for lung cancer, and smoking was only taken into account in five of the studies, while no association between alcohol consumption and lung cancer risk was seen in the case-control studies. In view of these results, IARC felt that there was "no indication that drinking of alcoholic beverages has a causal role in lung cancer".

References

1. International Agency for Research on Cancer (1988) Monographs on the evaluation of carcinogenic risks to humans. Volume 44: Alcohol drinking. IARC, Lyon.

5. Aluminium production

Table 5 gives details of the studies which investigated a possible association between working in aluminium production and the risk of lung cancer. The six standardized mortality ratios (SMR) given ranged from 114-174, with another study also finding a raised SMR but failing to give detailed results. Additionally, two estimates of relative risk were presented, both of which were above 1.00.

Workers in aluminium production are exposed to a wide variety of substances, frequently including aluminium compounds, carbon monoxide, chlorine and hydrogen chloride, coke, cryolite, fluorides, hydrogen fluoride, oil mists, pitch coal-tar and petroleum, polynuclear aromatic compounds, sulphur dioxide, and, less commonly, ammonia, asbestos, copper and copper oxide, cyanides, ozone, phosphine, silica, sodium hydroxide and welding fumes [1]. Information on measurements of benzo(a)pyrene concentrations was given by two studies (Konstantinov and Kuz'minykh, Spinelli et al), but apart from this no other data was available on the type of exposures experienced by the subjects in any of the studies. Thus, it is very difficult to determine which substances the workers were actually exposed to, and the effects of any one substance also remain unclear.

It has also been suggested that some of the studies (Gibbs, Rockette and Arena, Andersen) may be flawed due to an inappropriate choice of reference population, and that if a more suitable group had been selected the associations reported would have been reduced [3].

Despite these drawbacks, the consistency of the results from the various studies suggests that there may be an association between employment in aluminium production and the risk of lung cancer. In 1984, IARC classified aluminium production as "probably" carcinogenic to humans [1], but with the availability of results from further studies the evidence for carcinogenicity was later considered "sufficient" [2].

Table 5: Estimates of relative risk/standardized mortality ratio for aluminium production

Study	Population	Relative risk (95% limits)
Konstantinov and Kuz'minykh (1971)[1]	USSR Soderberg process workers	>1.00
Konstantinov et al (1974)[1]	USSR Soderberg process workers	>100*
Milham (1979)[1]	Prebake plant workers	117*
Giovanazzi and D'Andrea (1981)[1]	Soderberg process plant workers	174(p>0.05)*
Andersen et al (1982)[2]	Norwegian aluminium workers	159(p<0.05)*[3]
Rockette and Arena (1983)[1]	White US horizontal-stud Soderberg process workers	162(p<0.05)*
Gibbs (1985)[2]	Canadian Soderberg/prebake workers	140(p<0.05)*
Mur et al (1987)[2]	French aluminium workers	114*[3]
Spinelli et al (1991)[2]	Canadian Soderberg process workers	1.10

* Standardized mortality ratio

1 From IARC (1984)

2 From Ronneberg and Langmark (1992)

3 Estimated from data given

References

1. International Agency for Research on Cancer (1984) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 34: Polynuclear aromatic compounds, part 3, industrial exposures in aluminium production, coal gasification, coke production, and iron and steel founding, 37-64. IARC, Lyon.

2. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: an updating of IARC monographs volumes 1 to 42, 89-91. IARC, Lyon.

3. Ronneberg A and Langmark F (1992) Epidemiologic evidence of cancer in aluminum reduction plant workers. Am J Ind Med, 22, 573-590.

6. Antimony

Table 6 gives details of the only study found which gave data relevant to an investigation of the possible association between lung cancer risk and exposure to antimony. A standardized incidence ratio of 128 was calculated.

The main drawback of this study was that no objective measurements of exposure had been made. Therefore, it was impossible to determine the level of exposure to antimony, and to which other substances the workers had been exposed. Possible confounding exposures were noted to include arsenic, asbestos, cadmium, trivalent chromium, copper, lead, manganese, nickel oxide, zinc selenite, and sulphuric and hydrofluoric acids [1]. As several of these are thought to be potential carcinogens failure to record exposure to them may have introduced bias into the study results. Also, with only one study giving relevant results it is not really possible to evaluate the carcinogenicity of antimony to humans at this point.

Table 6: Estimate of standardized incidence ratio for exposure to antimony

Study	Population	Standardized incidence ratio
Sankila et al (1990)	Finnish glass factory workers	128(99-162)[1]

References

1. Sankila R, Karjalainen S, Pukkala E et al (1990) Cancer risk among glass factory workers: An excess of lung cancer? Br J Ind Med, 47, 815-818.

7. Arsenic

A number of studies were found which contained information on the possible association between exposure to arsenic and risk of lung cancer. Table 7 summarizes the relevant details. Seventeen relative risks were presented, ranging from 1.00-10.00, 14 of which were above 1.00. Ten standardized mortality ratios, of between 110-1189, were also given. Additionally, one study estimated an incidence rate of lung cancer of 205.6/100 000 per year when compared to the general male population, one reported 7% of deaths in smelter workers being due to lung cancer compared to 2.2-2.7% of deaths in workers in other areas, and one study simply stated that no excess in mortality from lung cancer was seen, but did not give any detailed results.

All of the studies for which the original papers were available had attempted to measure arsenic exposure, either by categorizing various jobs according to exposure [3-5], or by relying on indices such as length length of time of exposure [6]. However, the accounts of studies reported reported in the publications by IARC did not contain enough information to ascertain whether or not measurement of exposure had taken place.

Additionally, it has been suggested that, depending on the nature of their work, subjects exposed to arsenic may also be exposed to various other substances which may themselves be potential carcinogens. These include sulphur dioxide, chromium, nickel, iron, cadmium, radon, polyaromatic hydrocarbons such as benzpyrene, and various ore dusts [3-6]. As few of the studies measured for the possible occurrence of these other substances the exact nature of the subjects' exposures cannot be determined and it is difficult to separate out the effects of any one potential carcinogen.

Despite these drawbacks, when evaluating the data concerning the possible association between exposure to arsenic and risk of lung cancer, IARC considered the evidence for human carcinogenicity to be "sufficient" [1,2].

Table 7: Estimates of relative risk/standardized mortality ratio for exposure to arsenic

Study	Population	Relative risk (95% limits)
Thiers et al (1967)[1]	French vineyard workers	>1.00
Pinto et al (1968)[2]	Male copper smelter workers	305*[3]
Lee and Fraumeni (1969)	US smelter workers	329($p<0.01$)*[3]
Osburn (1969)[2]	Rhodesian gold miners	205.6[4]
Nelson et al (1973)[2]	Residents in apple-growing area	No excess mortality seen
Milham and Strong (1974)[2]	Copper smelter workers	222($p<0.001$)*[3]
Ott et al (1974)[2]	Insecticide packers	3.2(2.0-5.0)[3,5]
Blot and Fraumeni (1975)[2]	US males living near copper, lead or zinc industries	112*
	Females	110*
Tokudome and Kuratsune (1976)[2]	Japanese copper smelter workers	1189*
Pershagen et al (1977)[2]	Swedish males living near smelter	250($p=0.002$)*[3]
Rencher et al (1977)[2]	Copper corporation workers	7% of deaths in smelter workers due to neoplasm of respiratory tract compared to 2.2-2.7% in workers in other areas ($p<0.05$).
Axelson et al (1978)[2]	Swedish copper smelter workers	4.6(2.2-9.6)
Mabuchi et al (1979)[2]	Male pesticide workers	168*
Enterline and Marsh (1980,1982)[1]	US copper smelter workers	>1.00
Mabuchi et al (1980)	US pesticide workers - males	265($p<0.05$)*
	Females	168($p<0.05$)*

Table 7 continued

Matanoski et al (1980)[2]	US residents near pesticide plant - males	4.00
	Females	1.00
Wall (1980)[1]	Swedish smelter workers	6.00-8.00
Greaves et al (1981)[1]	Residents near smelter	1.00
Matanoski et al (1981,1983)[1]	Residents near smelters and pesticide plants	2.0-2.5
Pershagen et al (1981)	Swedish copper smelter workers	2.9(1.4-5.7)
Rom et al (1982)[1]	Residents near smelter	1.00
Luchtrath (1983)[1]	German vineyard workers	>1.00
Buiatti et al (1985)[1]	Italian hat makers	>1.00
Chen et al (1985,1986)[1]	Taiwanese residents	>1.00
Wingren and Axelson (1985)[1]	Swedish glass blowers	2.00
Lee-Feldstein (1986)[1]	Copper smelter workers	10.00
Wu et al (1989)	Chinese tin miners	3.7

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1987)

2 From International Agency for Research on Cancer (1980)

3 Tumours of respiratory system (ICD 6th rev. codes 160-164)

4 Incidence rate per 100 000 per year

5 Estimated from data given

References

1. International Agency for Research on Cancer (1980) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 23: Some metals and metallic compounds, 39-142. IARC, Lyon.

2. International Agency for Research on Cancer (1987) Monographs on the evaluation of the carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 100-106. IARC, Lyon.

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6. Wu K-G, Fu H, Mo C-Z, and Y L-Z (1989) Smelting, underground mining, smoking, and lung cancer: A case-control study in a tin mine area. Biomed Environ Sci, 2, 98-105.

8. Aryl hydrocarbon hydroxylase activity

Six studies were found which attempted to relate lung cancer risk to aryl hydrocarbon hydroxylase (AHH) activity, and details of these are given in Table 8. A relative risk of 6.0 was found for men with high AHH compared to those with low activity by one study, while another reported that 81% of lung cancer cases showed high AHH activity compared to only 31% of controls. Two other studies also noted increased AHH activity in lung cancer patients but did not give any results, while the remaining two found no differences between cases and controls.

It was suggested that AHH activity can be induced by the polycyclic hydrocarbons in tobacco smoke, thereby converting the hydrocarbons into carcinogenic metabolites [1-6], which may have accounted for the results observed by the studies. However, it is also possible the higher levels of AHH activity may somehow be caused by the development of lung cancer [5]. As all of the studies were of a case-control design, and collected data after the cases had been diagnosed with cancer, it is not clear whether this is so. Therefore, although the evidence presented in the table is suggestive of a positive relationship between lung cancer risk and high AHH activity, data from more studies are needed before more definite conclusions can be drawn.

Table 8: Estimates of relative risk for aryl hydrocarbon hydroxylase activity

Study	Population	Relative risk (95% limits)
Kellerman et al (1973)	US residents	Increased AHH activity in cases compared to controls ($p<0.001$)
McLemore et al (1981)	US hospital patients	81% cases and 31% controls showed high AHH activity ($p<0.001$)
Kouri et al (1982)	US hospital patients	Tendency for cases to show higher AHH activity than controls
Korsgaard et al (1984)	Swedish men	Relative risk of 6.0($p<0.05$) found for high AHH activity compared to low activity

Table 8 continued

Karki et al (1987)	Finnish residents	AHH activity similar in cases and controls
Bartsch et al (1992)	Italian men	No difference in AHH activity found between cases and controls

References

1. Bartsch H, Petruzzelli S, De Flora S et al (1992) Carcinogen metabolism in human lung tissues and the effect of tobacco smoking: Results from a case-control multicenter study on lung cancer patients. Environ Health Perspec, 98, 119-124.

2. Karki NT, Pokela R, Nuutinen L and Pelkonen O (1987) Aryl hydrocarbon hydroxylase in lymphocytes and lung tissue from lung cancer patients and controls. Int J Cancer, 39, 565-570.

3. Kellerman G, Shaw CR and Lutyen-Kellerman M (1973) Aryl hydrocarbon hydroxylase inducibility and bronchogenic carcinoma. N Engl J Med, 289, 934-937.

4. Korsgaard R, Trell E, Simonsson BG et al (1984) Aryl hydrocarbon hydroxylase induction levels in patients with malignant tumors associated with smoking. J Cancer Res Clin Oncol, 108, 286-289.

5. Kouri RE, McKinney CE, Slomiany DJ et al (1982) Positive correlation between high aryl hydrocarbon hydroxylase activity and primary lung cancer as analyzed in cryopreserved lymphocytes. Cancer Res, 42, 5030-5037.

6. McLemore TL, Martin RR, Wray NP, Cantrell ET and Busbee DL (1981) Reassessment of the relationship between aryl hydrocarbon hydroxylase and lung cancer. Cancer, 48, 1438-1443.

9. Asbestos

A brief review of the literature concerning asbestos exposure and the risk of lung cancer (as distinct from mesothelioma) revealed 22 studies on the subject, although this is far from exhaustive. Some relevant details of the studies are given in Table 9. For the cohort studies the estimates of relative risk ranged from 0.85-10.17, with 14 out of 15 being above 1.00, and the standardized mortality ratios calculated were between 105 and 210. Estimates of relative risk from the five case-control studies ranged from 0.97-3.29, with all but one of them being above 1.00.

Many of the studies included measurements of the levels of asbestos dust or fibre concentrations workers in the various industries were exposed to. However, two studies [16,22] did not measure the intensity of exposure, relying instead on years of employment as an index. Additionally, several of the studies [3,4,7,9,21,23] failed to make any objective measurement of the level of asbestos exposure, simply categorizing the respondents as to whether exposure had taken place or not. A failure to adequately measure exposure levels could obviously introduce inaccuracy into these studies.

Despite this limitation, the results of the studies were remarkably consistent, both for specific types of asbestos, and for those studies in which the type was not specified. This is reflected by IARC's description of the data relating asbestos exposure to risk of cancer in humans as "sufficient" (Group 1). It was also noted that there may be a synergistic effect with smoking, with the most likely model appearing to be multiplicative [11].

Table 9: Estimates of relative risk/standardized mortality ratio for asbestos exposure in men

Study	Population	Asbestos type	Relative risk (95% limits)
Cohort studies:			
Selikoff (1964)	New York/New Jersey insulation workers	NS	6.81[1]
Knox et al (1968)	Rochdale factory workers	CH, CR	10.17($p < 0.001$)[1]
McDonald et al (1971)	Quebec miners and millers	CH	9.9[2]
Gillam et al (1976)	South Dakota gold miners	AM	2.70 ($p < 0.05$)[3]
Hammond et al (1979)	US/Canadian insulation workers	NS	4.86 ($p < 0.001$)
Selikoff et al (1980)	New Jersey factory workers	AM	5.94[1]
Clemmesen and Hjalgrim-Jensen (1981)	Aalborg factory workers	NS	1.72[1]
McDonald et al (1982)	Pennsylvanian factory workers	CH, AM,CR	105.0*[3]
McDonald et al (1983)	South Carolina factory workers	CH	199.5*[3]
Acheson et al (1984)	London factory workers	AM	210 ($p < 0.01$)*
Newhouse et al (1985)	London factory workers	CR,	2.56[1]
		AM,CH	8.23[1,4]
	Laggers		3.6 ($p < 0.001$)
Albin et al (1990)	Swedish cement workers	CH, CR,AM	1.80(0.90-3.70)
Neuberger and Kundi (1990)	Vocklabruck cement workers	CH, CR	1.72(1.21-2.57)
Piolatto et al (1990)	Balangero asbestos miners	CH	1.10 ($p > 0.05$)
Hughes and Weill (1991)	New Orleans cement workers	NS	169 ($p < 0.01$)*
Sanden et al (1992)	Gothenburg shipyard workers	CH	0.85(0.53-1.30)
Raffn et al (1993)	Aalborg cement workers	CH, CR,AM	1.82(1.48-2.20)

Table 9 continued

Case-control studies:			
Blot et al (1980)	Virginian males not employed in shipyards	NS	2.20(1.10-4.60)
Blot et al (1982)	US shipyard workers	NS	3.17(0.87-11.5)[1]
	US construction workers		3.29(1.07-10.1)[1]
Garshick et al (1987)	US railroad workers	NS	0.97(0.83-1.14)
De Klerk et al (1991)	Wittenoom asbestos miners	CR	2.77(1.37-5.57)[1]
Minowa et al (1991)	Yokusuka shipyard workers	NS	2.54($p < 0.05$)

AM = Amosite; CH = Chrysotile; CR = Crocidolite; NS = Not specified

* Standardized mortality ratio

1 Estimated from data given

2 Death rate per 1000 men

3 Respiratory cancers (ICD codes 160-4)

4 Females only

References

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15. McDonald JC, McDonald AD, Gibbs GW, Siemiatycki J and Rossiter CE (1971) Mortality in the chrysotile asbestos mines and mills of Quebec. Arch Environ Health, 22, 677-686.

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22. Selikoff IJ (1964) Asbestos exposure and neoplasia. JAMA, 188, 22-26.

23. Selikoff IJ, Seidman H and Hammond EC (1980) Mortality effects of cigarette smoking among amosite asbestos factory workers. JNCI, 65, 507-513.

10. Atomic bomb explosions

Only two studies were found which gave information relevant to an investigation of lung cancer risk in relation to exposure to atomic bomb explosions, and details of these are shown in Table 10. One study estimated a relative risk of 3.55 while the other calculated a standardized mortality ratio of 21. Neither study gave any information on the significance of their findings.

With so few studies reporting it is not possible to fully evaluate the potential carcinogenicity of exposure to atomic bomb explosions.

Table 10: Estimates of relative risk/standardized mortality ratio for exposure to atomic bomb explosions

Study	Population	Relative risk
Knox et al (1983)	British servicemen	21*[1]
Prentice et al (1983)	Japanese residents	3.55

* Standardized mortality ratio

1 Tumours of respiratory system

References

1. Knox EG, Sorahan T and Stewart A (1983) Cancer following nuclear weapons tests. The Lancet, 815.

2. Prentice RL, Yoshimoto Y and Mason MW (1983) Relationship of cigarette smoking and radiation exposure to cancer mortality in Hiroshima and Nagasaki. JNCI, 70, 611-622.

11. Bakers/pastry cooks

Only two studies were found which looked at the possible association between employment as a baker/pastry cook and the risk of lung cancer, and these are shown in Table 11. Two standardized mortality ratios, of 87 and 131, were given, along with a mortality rate of 3.2 per 1000 per year, although it was not stated what reference group this rate was based on.

No information was available on the length of time the subjects had spent employed as bakers/pastry cooks, nor were any data on possible exposures presented. Furthermore, it was suggested that the raised mortality ratio calculated from the data presented by OPCS represented at best a weak association, but was more likely to be due to chance [2].

Table 11: Estimates of standardized mortality ratio for employment as a baker/pastry cook

Study	Population	Standardized mortality ratio
Boucot et al (1972)	US bakers/pastry cooks/chefs/cooks	3.2[1]
OPCS (1986)	UK foreman bakers/flour confectioners	87
	Bakers/flour confectioners	131($p<0.05$)

1 Per 1000 per year

References

1. Boucot KR, Weiss W, Seidman H, Carnahan W and Cooper DA (1972) The Philadelphia pulmonary neoplasm research project: basic risk factors of lung cancer in older men. Am J Epidemiol, 95, 4-16.

2. Office of Population Censuses and Surveys (1986) Occupational mortality: The Registrar General's decennial supplement for Great Britain, 1979-80, 1982-83. HMSO, London.

12. Barbers and hairdressers

Details of the studies which attempted to relate lung cancer risk to working as a barber, hairdresser or related occupation are given in Table 12. Eighteen relative risks were estimated, ranging from 0.5-6.0, with 16 being above 1.00. Ten standardized mortality ratios, of between 90-266, were also calculated, of which seven were raised. One study presented a standardized incidence ratio of 200, while another gave a proportional incidence ratio of 144.

Hairdressers may be exposed to over 5000 different chemicals, used in the formulation of hair colouring preparations, cleansing and conditioning products, preparations for hair styling, permanent waving and hair straightening, nail and skin products, and other products. However, none of the studies had made objective measurements of the subjects' exposures, and only the study by Guberan gave some limited details of products and chemicals respondents may have been exposed to. Thus, it is not possible to determine which chemicals the subjects were exposed to or the potential carcinogenicity of any one of them.

Therefore, IARC felt that there was "limited" evidence that occupation as a hairdresser or barber entails exposures that are carcinogenic [1].

Table 12: Estimates of relative risk/standardized mortality ratio for working as a barber/hairdresser

Study	Population	Relative risk (95% limits)
Registrar General (1958)[1]	UK male barbers/hairdressers	115*
	Female hairdressers/manicurists	200*
Garfinkel et al (1977)[1]	US female beauticians	6.0(p=0.06)
Menck et al (1977)[1]	US female beauticians	200(p<0.05)[2]
Alderson (1980)[1]	UK male hairdressers	1.02
Dubrow and Wegman (1982, 1983,1984)[1]	US male barbers	1.34
Logan (1982)	UK male hairdressers 1931	90*
	1951	115*
	1961	96*
	1971	115*
	Married women[3] 1951	90*
	1961	107*
	1971	111*
Kono et al (1983)[1]	Japanese female beauticians	1.21
Teta et al (1984)[1]	US female cosmetologists	1.41(p<0.05)
Guberan et al (1985)[1]	Swiss female hairdressers	1.9
	Male hairdressers	0.78
OPCS (1986)[1]	UK male barbers	266*[4]
Osorio et al (1986)[1]	US female cosmetologists	144[5]
Pearce and Howard (1986)[1]	New Zealand men	2.54
Malker et al (1987)[1]/Skov et al (1990)[1]	Swedish male hairdressers	1.5(1.2-1.8)
	Female hairdressers	1.6(1.1-2.2)
Lynge and Thygesen (1988)[1]/ Skov et al (1990)[1]/Skov and Lynge (1991)[1]	Danish female hairdressers	1.1
	Male hairdressers/barbers	1.1
Skov et al (1990)[1]	Norwegian female hairdressers	1.4
	Male hairdressers	1.6(p<0.05)
	Finnish female hairdressers	0.5
	Male hairdressers/barbers	1.5

Table 12 continued

Hrubec et al (1992)[1]	US male barbers/beauticians/ manicurists	1.6($p<0.05$)
Pukkala et al (1992)[1]	Finnish female hairdressers	1.72

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1993)

2 Standardized incidence ratio

3 According to husband's occupation

4 Estimated from data given

5 Proportional incidence ratio

References

1. International Agency for Research on Cancer (1993) Monographs on the evaluation of carcinogenic risks to humans. Volume 57: Occupational exposures of hairdressers and barbers and personal use of hair colourants; some hair dyes, cosmetic colourants, industrial dyestuffs and aromatic amines, 43-118. IARC, Lyon.

2. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. IARC, Lyon and OPCS, London.

13. Barbiturates

Only three studies could be found which contained data on a possible association between lung cancer and exposure to barbiturates, and details of these are given in Table 13. Two relative risks, of 1.3 and 1.4, and one standardized mortality ratio, of 173, were given.

While the study by Friedman recorded the use of three barbiturates, pentobarbital sodium, phenobarbital and secobarbital, separately, the other two studies appear to have only considered phenobarbital, and no details of exposures to other medication have been given. Therefore, it is possible that the subjects in these studies may be exposed to other drugs which may themselves have a carcinogenic effect.

Not surprisingly, IARC considered the evidence for the carcinogenicity of phenobarbital to humans to be "inadequate" [2]. Evaluations of the carcinogenicity of pentobarbital sodium and secobarbital were not made.

Table 13: Estimates of relative risk/standardized mortality ratio for exposure to barbiturates

Study	Population	Relative risk (95% limits)
White et al (1979)[1]	Epileptics	1.4(0.9-2.1)
Clemmesen and Hjalgrim-Jensen (1981)[1]	Epileptics	1.3(1.0-1.6)
Friedman (1981)	San Francisco outpatients	173(p<0.002)*

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1987)

References

1. Friedman GD (1981) Barbiturates and lung cancer in humans. JNCI, 67, 291-5.

2. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: an updating of IARC Monographs Volumes 1 to 42, 313-316. IARC, Lyon.

14. Barmen

The two studies found which attempted to relate lung cancer risk to employment as a barman are detailed in Table 14. Five standardized mortality ratios were calculated, lying in the range 100-194, of which four were raised. Two proportional mortality ratios, of 98 and 114, were also given, along with two proportional registration ratios, of 132 and 148.

Although the data presented in the table is consistent with an increased risk of lung cancer in barmen, it should be remembered that the data come from just two studies, both of which were based on mortality/morbidity statistics and no information on possible exposures, such as to alcohol and tobacco, is available. Elsewhere it has been suggested that barmen may be more exposed to tobacco than the general population, partly through environmental tobacco smoke exposure during their work, and partly through their own smoking habits [1]. However, the lack of data available makes it impossible to comment further on this theory.

Table 14: Estimates of standardized mortality ratio for employment as a barman

Study	Population	Standardized mortality ratio
OPCS (1978)	English/Welsh men, aged 15-64	114*
	Aged 65-74	98*
	Incidence 1966-7	148($p<0.01$)[1]
	1968-9	132($p<0.05$)[1]
Logan (1982)	English/Welsh men, 1951	117
	1961	137
	1971	165
	Married women[2], 1961	100
	1971	194

Footnote to Table 14

* Proportional mortality ratio

1 Proportional registration ratio

2 According to husband's occupation

References

1. Kjaerheim K and Andersen A (1993) Incidence of cancer among male waiters and cooks: Two Norwegian cohorts. Cancer Causes and Control, 4, 419-426.

2. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London and IARC, Lyon.

3. Office of Population Censuses and Surveys (1978) Occupational mortality: The Registrar General's decennial supplement for England and Wales, 1970-72. HMSO, London.

15. BCME/CMME

Only seven studies were found which looked at the risks of lung cancer after exposure to chloromethyl methyl ether (CCME) and/or bischloromethyl ether (BCME), and details of these are given in Table 15. The relative risks presented were all above 1.00, and ranged from 2.02-7.27.

Although the studies were concerned with workers exposed to CMME, the technical grade of this chemical, as used in the factories under observation, is nearly always contaminated with about 1-8% of BCME [1-4,6-11], so that workers will almost certainly have been exposed to both substances. Most of the studies recorded the length of exposure and made a subjective ranking of the amount of exposure, but as objective measurements of workroom air concentrations were taken by only one of the studies [4], the exact nature of the workers' exposure is mostly unknown.

Animal studies have suggested that the carcinogenic properties of BCME are far stronger than those of CMME [1,4,6,7] and one laboratory remarked that it was "one of the most potent carcinogens ever tested" [2]. It is not altogether surprising, then, that IARC felt there was "sufficient" evidence of the carcinogenic effects of BCME/CMME in humans, although the effects of the two compounds were not discussed separately [5].

Table 15: Estimates of relative risk for BCME/CMME exposure

Study	Population	Relative risk (95% limits)
Weiss et al (1979)	Philadelphia male factory workers	3.50 (1.82-6.75)
Weiss (1982)	Philadelphia male factory workers	2.91 (0.62-13.5)
McCallum et al (1983)	UK male factory workers	2.02 (0.83-4.90)
Zagraniski (1983)	Connecticut males	7.27 ($p > 0.05$)
Collingwood et al (1987)	US chemical workers	2.08 (1.36-3.16)
Maher and DeFonso (1987)	Philadelphia male factory workers	3.80 (2.24-6.46)
Gowers et al (1993)	French male factory workers	5.00 (2.00-12.3)

References

1. Collingwood KW, Pasternack BS and Shore RE (1987) An industry-wide study of respiratory cancer in chemical workers exposed to chloromethyl ethers. JNCI, 78, 1127-36.

2. Epstein SE (19??) The politics of cancer, 113-122. Sierra Club Books. (Incomplete reference)

3. Figueroa WG, Raszkowski R and Weiss W (1973) Lung cancer in chloromethyl methyl ether workers. N Engl J Med, 288, 1096-1097.

4. Gowers DS, DeFonso LR, Schaffer P et al (1993) Incidence of respiratory cancer among workers exposed to choromethyl-ethers. Am J Epidemiol, 137, 31-42.

5. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: an updating of IARC monographs volumes 1 to 42, 131-133. IARC, Lyon.

6. Maher KV and DeFonso LR (1987) Respiratory cancer among chloromethyl ether workers. JNCI, 78, 839-43.

7. McCallum RI, Woolley v and Petrie A (1983) Lung cancer associated with chloromethyl methyl ether manufacture: an investigation at two factories in the United Kingdom. Brit J Ind Med, 40, 384-9.

8. Travenius SZM (1982) Formation and occurrence of bis(chloromethyl)ether and its prevention in the chemical industry. Scand J Work Environ Health, 8 (Suppl 3), 1-86.

9. Weiss W (1976) Chloromethyl ethers, cigarettes, cough and cancer. J Occup Med, 18, 194-199.

10. Weiss W (1982) Epidemic curve of respiratory cancer due to chloromethyl ethers. JNCI, 69, 1265-70.

11. Weiss W, Moser RL and Auerbach O (1979) Lung cancer in chloromethyl ether workers. Am Rev Resp Dis, 120, 1031-7.

12. Zagraniski RT (1983) The role of occupation in the etiology of laryngeal carcinoma: a case-controlled study. Dis Abstr Int, 43, 3933.

16. Benzo(a)pyrene

It has been suggested that benzo(a)pyrene, a polycyclic aromatic hydrocarbon, may be responsible for the carcinogenic effects of soots, tars and oils observed in workers occupationally exposed to coal-soot, coal-tars and pitches, coal-tar fumes and some impure mineral oils, for whom excesses of lung cancer have been reported. In animal experiments benzo(a)pyrene has been shown to cause lung tumours, but assessment of the risk to humans is more difficult because subjects are also exposed to mixtures of other compounds, including other polycyclic aromatic hydrocarbons. Therefore, although several studies have measured benzo(a)pyrene exposure as an indication of exposure to soots, tars and oils, IARC considered the epidemiological data inadequate to evaluate the carcinogenicity to humans of benzo(a)pyrene itself [1,2].

References

1. International Agency for Research on Cancer (1973) Monographs on the evaluation of carcinogenic risk of chemicals to humans. Volume 3: Certain polycyclic aromatic hydrocarbons and heterocyclic compounds. IARC, Lyon.

2. International Agency for Research on Cancer (1974) Monographs on the evaluation of carcinogenic risk of chemicals to humans. Volume 4: Some aromatic amines, hydrazine and related substances, N-nitroso compounds and miscellaneous alkylating agents. IARC, Lyon.

17. Beryllium

Only six studies were found which provided data on a possible association between exposure to beryllium and the risk of lung cancer, and details of them are given in Table 17. Five standardized mortality ratios, of between 137 and 212 were given, along with two estimates of relative risk, of 2.00 and 1.26.

Several of the studies indicated that beryllium may show a carcinogenic effect after a short exposure period and a long latent period [1], and it was suggested that one explanation of this finding is that past short exposures, particularly those up to about 1950, may have entailed much higher accumulated doses than did long exposures in more recent periods, when airborne levels of beryllium became much lower [2]. However, as no objective measurements of beryllium exposure were available the total dose of beryllium received by the study subjects cannot be ascertained.

More importantly, when evaluating the evidence for carcinogenicity of beryllium and beryllium compounds, it was noted that two beryllium plants provided the database for all four of the studies cited by IARC [1]. Furthermore, the study by Ward et al also draws data from these two plants, along with five other plants, and as the study by Steenland and Ward is a continuation of that by Infante et al, it can be said that none of the studies is totally independent.

Not surprisingly then, IARC considered the evidence for the carcinogenicity of beryllium in humans to be "limited", although it was stated that "beryllium should be considered suspect of being carcinogenic to humans" [1].

Table 17: Estimates of relative risk/standardized mortality ratio for exposure to beryllium

Study	Population	Relative risk (95% limits)
Infante et al (1980)[1]	US registry data	212*
Mancuso (1980a)[1]	US beryllium workers - Ohio	199(p<0.01)*
	Pennsylvania	137*
Mancuso (1980b)[1]	US beryllium workers	140(p<0.01)*
Wagoner et al (1980)[1]	US beryllium workers	137(p<0.05)*
Steenland and Ward (1991)	US registry data	2.00(1.33-2.89)
Ward et al (1992)	US beryllium workers	1.26(1.12-1.42)

* Standardized mortality ratio

1 From International Agency for Research on Cancer (1980)

References

1. International Agency for Research on Cancer (1980) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 23: Some metals and metallic compounds, 143-204. IARC, Lyon.

2. Saracci R (1991) Beryllium and lung cancer: adding another piece to the puzzle of epidemiologic evidence. JNCI, 83, 1362-1363.

3. Steenland K and Ward E (1991) Lung cancer incidence among patients with beryllium disease: A cohort mortality study. JNCI, 83, 1380-1385.

4. Ward E, Okun A, Ruder A, Fingerhut M and Steenland K (1992) A mortality study of workers at seven beryllium processing plants. Am J Ind Med, 22, 885-904.

18. Bitumens

Only three studies attempted to relate lung cancer risk to exposure to bitumens, and details of these are given in Table 18. Five standardized mortality ratios, ranging from 92-496, were calculated, of which four were raised. In addition, one study presented a proportional mortality ratio of 161.

It was noted that in the USA most roofers work with both bitumens and coal tar pitches [1], but as no information was available from the studies on the exposures the workers were subjected to it is not possible to attribute the excess cancer risk to either one or the other substance. Therefore, IARC classified the evidence for the carcinogenicity of bitumens to humans as "inadequate" [2].

Table 18: Estimates of standardized mortality ratio for exposure to bitumens

Study	Population	Standardized mortality ratio
Hammond et al (1976)[1]	US roofers, 9-19 years union membership	92
	20-29 years	152
	30-39 years	150
	40+ years	247
Menck and Henderson (1976)[1]	US roofers	496
Milham (1982)[1]	US roofers and slaters	161[2]

1 From International Agency for Research on Cancer (1985)

2 Proportional mortality ratio

References

1. International Agency for Research on Cancer (1985) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 35: Polynuclear aromatic compounds, part 4, bitumens, coal-tars and derived products, shale-oils and soots, 39-81. IARC, Lyon.

2. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 133-134. IARC, Lyon.

19. Body height

Five studies provided data relevant to an investigation of the possible association between lung cancer risk and body height, and details of them are given in Table 19. Seven relative risks were estimated, ranging from 0.99-3.7, of which five were above 1.00. Incidence rates of between 8.8 and 19.0 per 1000 men were presented by one study. Finally, one study reported no apparent association between lung cancer risk and body height, but did not give any detailed results.

From the data presented in the table it can be seen that there is little consistent evidence of an association between lung cancer and body height, and therefore any risk, if it does exist, is probably very small.

Table 19: Estimates of relative risk for tall compared to short body height

Study	Population	Relative risk (95% limits)
Cochrane and Moore (1983)	UK men	No association seen between height and lung cancer risk
Lee and Kolonel (1983)	Hawaiian male smokers, highest tertile of height	3.7(1.2-11.5)
	Male non-smokers	2.9(0.6-13.0)
	Female smokers	1.8(0.6-5.2)
	Female non-smokers	3.4(0.5-24.1)
Nomura et al (1983)	Hawaiian men of Japanese descent, 140-57cm	14.0[1]
	160cm	8.8[1]
	163cm	14.7[1]
	165-68cm	13.4[1]
	170-88cm	19.0[1]

Table 19 continued

Wynder and Goodman (1983)	White men	Relative risk of 1.00(0.98-1.01) found for height in multiple regression
	Women	Relative risk of 0.99(0.97-1.02) found for height in multiple regression
Knekt et al (1991)	Finnish men, >178cm tall compared to ≤169cm tall	1.2(0.6-2.1)

1 Incidence per 1000 men

References

1. Cochrane AL and Moore F (1983) Body height and lung cancer risk. Lancet, May 21, 1162 (Letter).

2. Knekt P, Seppanen R, Jarvinen R et al (1991) Dietary cholesterol, fatty acids, and the risk of lung cancer among men. Nutr Cancer, 16, 267-275.

3. Lee J and Kolonel LN (1983) Body height and lung cancer risk. Lancet, April 16, 877 (Letter).

4. Nomura A, Heilbrun LK and Stemmerman GN (1983) Body height and lung cancer risk. Lancet, May 21, 1162 (Letter).

5. Wynder EL and Goodman MT (1983) Body height and lung cancer risk. Lancet, May 21, 1162-1163 (Letter).

20. Body mass index

Three studies attempted to relate the risk of lung cancer to body mass index, and details of them are given in Table 20. For low body mass index three relative risks, ranging from 1.7-2.3, were given. The one study which used low body mass index as the baseline estimated a relative risk of 0.5 for those with a high body mass index.

It is well known that the development of cancer often leads to a drop in body weight so that any observed association between lung cancer and low body mass index may be due to, rather than a cause of, the disease. The study by Kabat tried to get round this problem by asking respondents about their weight five years prior to diagnosis. However, it is not certain how accurate such self-reported estimates of weight will be. The studies by Knekt, which were of a prospective design, collected information on weight at the start of the follow-up period.

While the evidence presented in the table suggests an association between lung cancer risk and low body mass index, with so few studies reporting it is not really possible to evaluate the relationship properly.

Table 20: Estimates of relative risk for body mass index

Study	Population	Relative risk (95% limits)
Low body mass index:		
Kabat (1991)	US men, lowest compared to highest quartile	1.7(1.4-2.1)
	Women	2.3(1.8-3.2)
Knekt et al (1991a)	Finnish men, lowest compared to highest quartile	1.8
High body mass index:		
Knekt et al (1991b)	Finnish men, highest compared to lowest quartile	0.5(0.3-0.9)

References

1. Kabat GC (1991) Body mass index and lung cancer risk. Am J Epidemiol, 134, 725 (Abstract).

2. Knekt P, Heliovaara M, Rissanen A et al (1991a) Leanness and lung cancer risk. Int J Cancer, 49, 208-213.

3. Knekt P, Seppanen R, Jarvinen R et al (1991b) Dietary cholesterol, fatty acids, and the risk of lung cancer among men. Nutr Cancer, 16, 267-275.

21. Brewers

Only two studies were found which gave any information on the possible association between lung cancer risk and brewers, and details are given in Table 21. Four standardized mortality ratios were presented, ranging from 108-149.

No data was available on possible exposures brewers might be subjected to, and no hypotheses were put forward to explain the observed increase in lung cancer risk. With so little information available, it is not really possible to evaluate the potential carcinogenicity of employment as a brewer.

Table 21: Estimates of standardized mortality ratio for brewers

Study	Population	Standardized mortality ratio
OPCS (1978)	English/Welsh brewers/wine makers	119
Logan (1982)	English/Welsh male brewers, 1951	149
	1961	108
	1971	142

References

1. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London and IARC, Lyon.

2. Office of Population Censuses and Surveys (1978) Occupational mortality: The Registrar General's decennial supplement for England and Wales, 1970-72. HMSO, London.

22. Bricklayers

Table 22 gives details of the four studies found which investigated a possible association between lung cancer risk and employment as a bricklayer. Seven standardized mortality ratios ranging from 96-147 were calculated, of which six were raised. Two proportional mortality ratios, of 111 and 113, were also presented, along with two proportional registration ratios, of 125 and 128, and one standardized mortality odds ratio of 143. One study estimated a relative risk of 2.1.

Although it was suggested that workers in the construction industry may be potentially exposed to asbestos [1,3], no attempts were made by the studies to measure the levels of this or any other possible carcinogen that the workers may have been subjected to. Thus, it is not possible to identify the agent or agents responsible for the increased risk of lung cancer observed.

Table 22: Estimates of standardized mortality ratio/relative risk for bricklayers

Study	Population	Standardized mortality ratio
OPCS (1978)	E/W male bricklayers/tile setters aged 15-64	111($p<0.05$)[1]
	Aged 65-74	113($p<0.05$)[1]
	Incidence 1966-7	125($p<0.01$)[2]
	1968-9	128($p<0.01$)[2]
Logan (1982)	E/W male bricklayers - 1931	104
	1951	113
	1961	136
	1971	147
	Married women[3] - 1951	100
	1961	96
	1971	123
Milne et al (1983)	US brickmasons	2.1*
Dubrow and Wegman (1984)	US brickmasons/stonemasons/tile setters	143($p<0.05$)[4]

Footnote to Table 22

E/W = English/Welsh

* Relative risk

1 Proportional mortality ratio

2 Proportional registration ratio

3 According to husband's occupation

4 Standardized mortality odds ratio

References

1. Dubrow R and Wegman DH (1984) Cancer and occupation in Massachusetts: A death certificate study. Am J Ind Med, 6, 207-230.

2. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London.

3. Milne KL, Sandler DP, Everson RB and Brown SM (1983) Lung cancer and occupation in Alameda County: A death certificate case-control study. Am J Ind Med, 4, 565-575.

4. Office of Population Censuses and Surveys (1978) Occupational mortality: The Registrar General's decennial supplement for England and Wales, 1970-72. HMSO, London.

23. Building labourers

Table 23 gives details of the studies found which investigated a possible association between lung cancer and employment as a building labourer. Eleven standardized mortality ratios were calculated, ranging from 97-175, of which 10 were raised. Four proportional mortality ratios were also presented, lying in the range 109-118, along with four proportional registration ratios, ranging from 82-122, of which three were raised. Seven relative risks were estimated, ranging from 1.18-2.5.

Although it was suggested by one of the studies that exposure to asbestos may be responsible for the increase in lung cancer risk observed among building labourers [4], no attempt was made by this, or any of the other studies to measure the workers' potential exposures. Thus, no evidence is available to either support or refute this hypothesis. Indeed, with such little information available it is difficult to interpret the results, but from the table it can be seen that any increase in risk, if it does exist, is probably not very large.

Table 23: Estimates of standardized mortality/relative risk ratio for building labourers

Study	Population	Relative risk (95% limits)
Blot et al (1978)[1]	US general construction workers	2.0
OPCS (1978)	E/W male building/contracting labourers aged 15-64	113($p<0.05$)[2]
	Aged 65-74	116($p<0.05$)[2]
	Incidence 1966-7	82[3]
	1968-9	122($p<0.01$)[3]
	Male general construction workers aged 15-64	118($p<0.05$)[2]
	Aged 65-74	109($p<0.05$)[2]
	Incidence 1966-7	114($p<0.01$)[3]
	1968-9	119($p<0.01$)[3]
Gottlieb et al (1979)[1]	US general construction workers	1.3($p>0.05$)

Table 23 continued

Blot et al (1982)[1]	US general construction workers	1.4
Logan (1982)	E/W men - 1931	153*
	1951	175*
	1961	124*
	1971	97*
	Married women[4] - 1951	163*
	1961	105*
	1971	135*
Blot et al (1983)[1]	US general construction workers	1.4(p>0.05)
Milne et al (1983)	US male construction labourers	1.9(p<0.05)
OPCS (1986)	UK male building/construction workers	131(p<0.01)*
	Married women[4]	134(p<0.01)*
	Male civil engineering/craftsmen's mates/other building labourers	135(p<0.01)*
	Married women[4]	131(p<0.05)*
Alavanja et al (1990)	US female housing construction workers	2.5(0.4-15.1)
Keller and Howe (1993)	US general construction workers	1.18(1.02-1.36)

E/W = English/Welsh

* Standardized mortality ratio

1 From Williams Pickle

2 Proportional mortality ratio

3 Proportional registration ratio

4 According to husband's occupation

References

1. Alavanja MCR, Brownson R and Boice JD (1990) Risk factors for lung cancer among nonsmoking women. Society for Epidemiologic Research, Snowbird, Utah, USA.

2. Keller JE and Howe HL (1993) Cancer in Illinois construction workers: A study. Am J Ind Med, 24, 223-230.

3. Logan WPD (1982) Cancer mortality by occupation and social class 1851-1971. HMSO, London.

4. Milne KL, Sandler DP, Everson RB and Brown SM (1983) Lung cancer and occupation in Alameda County: A death certificate case-control study. Am J Ind Med, 4, 565-575.

5. Office of Population Censuses and Surveys (1978) Occupational mortality: The Registrar General's decennial supplement for England and Wales, 1970-72. HMSO, London.

6. Office of Population Censuses and Surveys (1986) Occupational mortality: The Registrar General's decennial supplement for Great Britain, 1979-80, 1982-83. HMSO, London.

7. Williams Pickle L, Correa P and Fontham E (1984) Recent case-control studies of lung cancer in the United States. In: Lung cancer: Causes and prevention, 101-115. Verlag Chemie International Inc.

24. Butchers

Table 24 summarizes the available evidence on lung cancer risk for individuals employed as butchers, or in associated jobs in the meat industry. Seventeen standardized mortality ratios were presented, ranging from 85-331, with 14 being above 100. Three standardized incidence ratios, ranging between 120-178, were also given, along with one proportional mortality ratio of 154 and one relative risk estimate, of 1.17. Additionally, one study reported finding 36 cases of lung cancer in butchers compared to only 15 cases in a comparison population of bakers.

The consistent excess of lung cancer amongst butchers and those working in related jobs in the meat industry suggests that there may indeed be some risk involved, but no plausible mechanism has yet been suggested. One theory is that butchers and workers in slaughterhouses are exposed to various animal viruses, such as the bovine leukaemia virus, the avian leukosis viruses and Marek's disease virus, which are known to cause cancer in cows and chickens, and may also increase the risk of cancer in humans [4]. Another possibility, stemming from the observation of an unusually high prevalence of viral warts in butchers, is that human wart viruses may also be capable of producing malignant changes, and thus may be responsible for the excess of lung cancers in butchers [8]. It has also been suggested that risks in more recently exposed workers could be related to new exposures in the meat industry such as fumes from heated polymer wraps [9]. One further possibility, not discussed by any of the studies, is that butchers may consume more red meat than average and thus have a higher intake of dietary fat, and this may have a contributory role in the risk of developing lung cancer.

Table 24: Estimates of standardized mortality ratio/relative risk for butchers

Study	Population	Standardized mortality ratio
Doerken and Rehpenning (1982)	German butchers	36 lung cancer cases in butchers compared to 15 in bakers ($p<0.01$)
Fox et al (1982)	English/Welsh male butchers	116
	Male butchers 1966-67	127 ($p<0.01$)[1]
	Male butchers 1968-70	120 ($p<0.01$)[1]
	Danish butchers in slaughterhouses	253
	Butchers working elsewhere	165
	Unskilled workers in slaughterhouses	85
	Swedish butchers in slaughterhouses	178[1]
	Butchers working elsewhere	130
	Other employees in slaughterhouses	148
Griffith (1982)	English/Welsh butchers	127
	Meat and fish curers and smokers	200
	Slaughterhouse workers	131
	Proprietors of retail businesses for sale of foods, including meat	151
Johnson and Fischman (1982)	US butchers	154[2]
Milham (1982)	US butchers	99
Vena et al (1982)	US employees in meat industry	1.17(0.67-2.05)*
Lynge et al (1983)	Danish self-employed butcher's shop employees	169
	Skilled butcher's shop employees	148
	Skilled slaughterhouse workers	159
	Unskilled slaughterhouse workers	100
OPCS (1986)	Great Britain - foreman butchers	331 ($p < 0.01$)
	butchers	176 ($p < 0.01$)

Footnote to Table 24

* Relative risk

1 Standardized incidence ratio

2 Proportional mortality ratio

References

1. Doerken H and Rehpenning W (1982) Lung cancer in butchers. The Lancet, Mar 6, 561. (Letter)

2. Fox AJ, Lynge E, Malker H (1982) Lung cancer in butchers. The Lancet, Jan 16, 165-166. (Letter)

3. Griffith GW (1982) Lung cancer in butchers. The Lancet, Feb 13, 399. (Letter)

4. Johnson ES and Fischman HR (1982) Cancer mortality among butchers and slaughterhouse workers. The Lancet, Apr 17, 913-914. (Letter)

5. Lynge E, Andersen O and Kristensen TS (1983) Lung cancer in Danish butchers. The Lancet, Mar 5, 527-528. (Letter)

6. Milham S (1982) Lung cancer in butchers. The Lancet, Mar 20, 690. (Letter)

7. Office of Population Censuses and Surveys (1986) Occupational mortality: the Registrar General's decennial supplement for Great Britain, 1979-80, 1982-83, 81-82. HMSO, London.

8. Pegum JS (1982) Lung cancer in butchers. The Lancet, Mar 6, 561. (Letter)

9. Vena JE, Byers T, Swanson M and Cookfair D (1982) Lung cancer in butchers? The Lancet, 2, 713. (Letter)

25. Cadmium

Ten studies were found which gave information relevant to an investigation of the possible association between lung cancer risk and exposure to cadmium, and details are given in Table 25. The relative risks estimated ranged from 0.26-17.0, with 10 out of 12 being above 1.00.

Seven studies (Ding, Elinder, Holden, Kjellstrom, Lemen, Sorahan, Stayner) took objective measurements of the levels of cadmium workers were exposed to, while the study by Kazantzis et al grouped respondents by estimated exposure level. Additionally, four studies (Ding, Lemen, Kazantzis, Sorahan) recorded the duration of exposure. However, it was noted that in some studies workers may also have been exposed to other substances, particularly nickel compounds, but also arsenic, beryllium, chromium, indium, lead, thallium and emissions from a variety of heated mineral oils [3,4], although no measurements of these appear to have been taken. Therefore, it is possible that exposure to one or more of these substances may have produced the observed results.

When initially evaluating the carcinogenicity of cadmium to humans in 1976, IARC felt the evidence to be "limited" [3], while a review of occupational exposures by Blot described cadmium as only a "possible" carcinogen [1]. However, by 1993, IARC concluded there was "sufficient" evidence that cadmium and cadmium compounds "are carcinogenic to humans" [4].

Table 25: Estimates of relative risk for cadmium exposure

Study	Population	Relative risk (95% limits)
Lemen et al (1976)[1]	US smelter workers	2.35[2]
Kjellstrom et al (1979)[3]	Swedish battery workers	1.48(0.17-5.35)
Kazantzis et al (1992)[3]/ Kazantzis and Blanks (1992)[3]	UK cadmium workers	1.12(1.00-1.24)
Elinder et al (1985)[3]	Swedish battery workers	1.33(0.57-2.62)
Kipling and Waterhouse (1967)[3]	UK battery workers	1.14(0.37-2.65)
Sorahan (1987)[3]	UK battery workers	1.30(1.07-1.57)
Holden (1980)[3]	UK copper-cadmium alloy factory workers, urban	1.78(0.77-3.50)
	Rural	0.26(0.03-0.92)
Stayner et al (1992)[3]	US cadmium recovery workers	1.49(0.95-2.21)
Ding et al (1987)[3]	Chinese smelter workers, cadmium shop	6.65
	Sintering shop	17.0
Siemiatycki (1991)[3]	Canadian men	1.00

1 From International Agency for Research on Cancer (1976)

2 Estimated from data given

3 From International Agency for Research on Cancer (1993)

References

1. Blot WJ (1984) Lung cancer and occupational exposures. In: Lung cancer: Causes and prevention, 47-64. Verlag Chemie International Inc.

2. International Agency for Research on Cancer (1976) Monographs on the evaluation of carcinogenic risk of chemicals to man. Volume 11: Cadmium, nickel, some epoxides, miscellaneous industrial chemicals and general considerations on volatile anaesthetics, 39-74. IARC, Lyon.

3. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 139-142.

4. International Agency for Research on Cancer (1993) Monographs on the evaluation of carcinogenic risks to humans. Volume 58: Beryllium, cadmium, mercury, and exposures in the glass manufacturing industry, 119-237. IARC, Lyon.

26. Carbon-blacks

Table 26 gives details of the 25 studies found which investigated the risk of lung cancer in workers potentially exposed to carbon-blacks. The standardized mortality ratios calculated ranged from 47-434, with 17 out of 25 being raised. Five relative risks were also estimated, ranging from 1.4-2.3. Additionally, one study reported a mortality rate of 1.07 per 1000 per year, while another calculated a standardized incidence ratio of 126.

Exposure to carbon-blacks occurs mostly in the rubber and printing industries, and such workers are likely to be exposed to a variety of other chemicals, many of which have been shown experimentally to be mutagenic or carcinogenic. These include mineral oils, curing fumes, some monomers, solvents, nitroso compounds and aromatic amines, thiurams and dithiocarbamate compounds, ethylenethiourea, di(2-ethylhexyl)phthalate, di(2-ethylhexyl)adipate and hydrogen peroxide [2]. However, due to a lack of historical industrial hygiene data none of the studies could accurately ascertain exposure, although some of the studies of rubber workers (Baxter and Werner, Delzell, Fox, McMichael 1976, Monson and Fine, Monson and Nakano 1976b, Parkes, Zhang) did attempt to classify workers into particular job categories as a substitute for exposure categories. Thus, attempting to evaluate the potential carcinogenicity of any one compound will be extremely difficult.

Therefore, IARC's evaluation of the evidence for the carcinogenicity to humans of carbon-blacks as "inadequate" [3,4] appears to be justified.

Table 26: Estimates of standardized mortality ratio/relative risk for exposure to carbon-blacks

Study	Population	Standardized mortality ratio
Goldstein et al (1970)[1]	US newspaper plant workers	1.07[2]
Greenberg (1972)[1]	UK newspaper printers	133($p<0.01$)[3]
Moss et al (1972)[1]/Moss (1973)[1]	UK newspaper workers	134($p<0.01$)[3]
Fox et al (1974)	British rubber workers	118
McMichael et al (1974)[4]	US rubber workers	83
Andjelkovich et al (1976)[4]	US rubber workers	83
Fox and Collier (1976)[4]	British rubber workers	127
McMichael et al (1976a,b)[4]	US Receivers/shippers	1.9*[5]
	Compounders/mixers	1.4*[5]
	Mill-mixers	2.1*[5]
	Extruders	1.4*[5]
	Reclaimers	2.3*[5]
Menck and Henderson (1976)[1]	US newspaper printers	98
Monson and Nakano (1976a)[4]	US rubber workers	92
Monson and Nakano (1976b)[4]	US female non-tyre rubber workers	333
Andjelkovich et al (1977)[4]	US workers exposed to synthetic latex	434
Lloyd et al (1977)[1]	US newspaper pressmen	112[3]
Andjelkovich et al (1978)[4]	US female rubber workers	191[3]
Monson and Fine (1978)[4]	US tyre curers	220[3]
	Tyre moulders	200[3]
	Fuel cell/deicer manufacturers	158[3]
Baxter and Werner (1980)[4]	British rubber workers	115[3]
Bovet and Lob (1980)[4]	Swiss rubber workers	47
Paganini-Hill et al (1980)[1]	US newspaper pressmen	149[3]
Robertson and Ingalls (1980)[1]	US carbon black workers	88[3]
Delzell and Monson (1981)[4]	US rubber workers	84
Kilpikari et al (1981)[4]	Finnish rubber workers	150[5]

Table 26 continued

Delzell et al (1982)[4]	US tyre workers - mortality	99
	Incidence	126[6]
Parkes et al (1982)[4]	British rubber workers	122
Hodgson and Jones (1985)[7]	UK carbon black workers	>100
Zhang et al (1989)	Chinese rubber workers	133

* Relative risk

1 From International Agency for Research on Cancer (1984)

2 Per 1000 per year

3 Estimated from data given

4 From IARC (1982)

5 Tumours of respiratory system

6 Standardized incidence ratio

7 From International Agency for Research on Cancer (1987)

References

1. Fox AJ, Lindars DC and Owen R (1974) A survey of occupational cancer in the rubber and cablemaking industries: Results of five-year analysis, 1967-71. Br J Ind Med, 31, 140-151.

2. International Agency for Research on Cancer (1982) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 28: The rubber industry. IARC, Lyon.

3. International Agency for Research on Cancer (1984) Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Volume 33: Polynuclear aromatic compounds, part 2, carbon blacks, mineral oils and some nitroarenes, 35-168. IARC, Lyon.

4. International Agency for Research on Cancer (1987) Monographs on the evaluation of carcinogenic risks to humans. Supplement 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42, 142-143 and 332-334. IARC, Lyon.

5. Zhang Z-F, Yu S-Z, Li W-X and Choi BCK (1989) Smoking, occupational exposure to rubber, and lung cancer. Br J Ind Med, 46, 12-15.